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The hyperventilation syndrome, first described by de Kosta in 1870 and observed in neurosis patients, consists of a marked increase of ventilation while at rest, without any organic changes on the part of the cardiovascular and respiratory systems. The cause of development of the hyperventilation syndrome lies in the enhanced excitability of the respiratory center. Most frequently a considerable deepening of respiration takes place during the hyperventilation syndrome; at times, however, it is very rapid and superficial, or, moreover, with a superficial and rapid respiration, there are observed very deep inspirations. Not infrequently, even at rest the thorax remains constantly in a somewhat inspiratory state; however, a complete expiration is always possible. The maximum ventilation of the lungs is generally diminished. Meili (1948) notes that, in contrast to healthy individuals, the oxygen inhalation during work does not reduce ventilation in the hyperventilation syndrome patients (Knipping, 1955).

The clinical picture of the hyperventilation syndrome is not clear-cut, because there are combined with it symptoms characteristic of neurosis. Hyperventilation per se leads to the increase of carbon dioxide elimination and, as a result, to hypocapnia. It is known that, under normal conditions, a marked hypocapnia causes apnea, as a result of the humoral regulation of respiration. Besides, experiments on dogs showed that apnea may take place also in hyperventilation with a mixture of 18 percent of oxygen and 5.5 percent of carbon dioxide, when there are no humoral changes in the blood whatsoever. In this case the apnea is of shorter duration than in air hyperventilation; still, it does take place,

which underlines the role of neural factors capable of inhibiting ventilation in cases of marked hyperventilation, independently of changes in blood gases (Binet, Strumza, 1953). In the hyperventilation syndrome functional impairments of the nervous system lead to the depression of both, the neural and humoral mechanisms, and no apnea takes place. Therefore, in the hyperventilation syndrome the characteristic feature is continuous hypocapnia which, though it is not so marked as to lead to a hyperventilation tetanus and pronounced alkalosis, still contributes to the emergencies of definite symptoms, in the first place, on the part of the cardiovascular system, and, in the second place, on the part of the nervous system. The patients experience a lack of air and difficulty in swallowing; as a result of enhanced ventilation and acceleration of fluid evaporation from the mucosa, there is a development of dryness in the mouth, and aerophagy and meteorism are observed; also, vertigo, paraesthesiae and hyperesthesiae, vague pain in the chest, noises in the head, tremor of the extremities, and sensation of muscle tightening. Hypocapnia may also lead to vascular dilation, to a decrease and, at times, increase of blood pressure (Rice, 1950); Hoff et al., 1952; Lewis, 1954). The clinical picture shows that this syndrome may appear under the guise of other disturbances (cardiovascular, nervous, pulmonary and intestinal). Frequently, hyperventilation may become fixated reflectorily, at first emerging upon the sensation of fear, or exertion, and, later, following any psychic load (Hoff, etc.).

It is important to point out that a simple visual observation of the patient does not always help to diagnose the hyperventilation syndrome, especially in the case of uniform regular breathing. Pneumography is a very important method of diagnosis if it is conducted on one level of the thorax. Therefore, the best method of diagnosis of the hyperventilation syndrome is a spirographic examination by means of an apparatus of a closed type (Knipping). The examination can also be conducted on the Krog apparatus.

We examined four patients manifesting the hyperventilation syndrome in the basal metabolism laboratory of the city hospital. The spirograms of air respiration (two and a half minutes) and oxygen respiration (five minutes) were recorded on a Krog apparatus. Then, by means of dry gas clocks the vital capacity of the lungs was determined four times and the maximum ventilation of the lungs -- twice (with intervals of a few minutes' between tests), and the maximal result was evaluated. The data obtained on vital capacity, maximum ventilation of the lungs, minute volume of respiration and absorption of oxygen were compared and ex-

pressed in percentages as related to the normal figures for the tested individual calculated according to the data of normal basal metabolism (A. G. Dembo, 1957).

Cited below are brief data on the observed patients.

1. Female patient, M. I., 18 years old, has complained for several months of nervousness, sweating, noises in the head and headaches. At times she noted attacks of marked weakness, sensation of dryness in the mouth, dyspnea when at rest, observed during states of agitation; without agitation, there was no dyspnea noted. Clinical and X-ray examination showed no pathological changes in the internal organs. Liability of the pulse was observed (100 beats per minutes when patient was in an upright position, 72 beats -- in a reclining position); arterial pressure: 120/72 to 125/80 mm of the mercury column.

2. Female patient, L. M., 50 years old, having the same complaints as the previous patient; in addition, for the past few years she has observed aerophagy, sensation of abdominal distension, and hand tremors. She was examined in the clinics of the city of Tartu; there were no organic changes observed in the cardiovascular and respiratory systems. Clinical and X-ray examination at the hospital elicited no pathology on the part of the internal organs. Pulse, with patient standing 80, reclining -- 72 beats per minute; arterial pressure 145/85 mm of mercury column. There was no tremor observed.

3. Female patient, M.V., 56 years old, has suffered for the past few years from a progressive lipodystrophy in the form of a lipohypertrophy of the lower half of the body, and hypertension of the first stage. The basic complaints were weakness, quick fatigue, dryness of the mouth, sensation of tightness in the muscles, vertigo, and paraesthesiae of the extremities. There was no dyspnea or headaches. On hospital examination there was no pathology elicited on the part of the organs of external respirations. Pulse -- 64 to 68 beats per minute. During the few weeks prior to the spirographic examination, the arterial pressure was never higher than 140/90 mm of mercury column.

4. Patient, B. P., 43 years old, had suffered since early childhoods from attacks of dyspnea which manifested themselves in the need of taking maximal deep inspirations every minute. In 1938 he was examined at the Hospital im Kuybyshev in Leningrad and no pathology of the cardiovascular and respiratory systems was elicited. A year prior to his admission to the hospital, there appeared weakness; nervousness and sweating increased somewhat, and a sensation of tremors of the hands and feet appeared. The dyspnea

was most annoying when the patient was lying on his back, and least of all -- when lying on his back, but with his head bent backward (!). Cooling off, or various odors had no effect on the dyspnea. The patient has been coming repeatedly to various therapeutic establishments complaining of dyspnea; diagnoses were made of bronchial asthma, or myocardial dystrophy (?). Clinical examination showed tremor of the fingers, liability of pulse (standing -- 102, lying -- 68 beats per minute), arterial pressure: 105/80 mm mercury column. Clinical and X-ray examination elicited no changes in the internal organs.

As seen from the above, the patients had various complaints: general weakness, sensations of tremors, aerophagy, etc.; tremor and liability of pulse were noted. No clinical changes in internal organs were observed; roentgenoscopy of the thorax, analysis of the blood, urine and feces, and basal metabolism were normal. There were no clinical symptoms of the pathology of endocrine glands. Neurological examination (Docent V. Ya. Yuprus) elicited in all patients a neurosis of the neurasthenic type and the absence of organic changes of the nervous system. All patients were kept under continuous observation in a city hospital, and the observation which lasted many months confirmed the absence of organic diseases of the cardiovascular and respiratory system.

Patients with a hyperventilation syndrome: M. I., L. M., and M. V. (females) complained of no dyspnea even while at work (except the agitation dyspnea in patient M. I.); a marked enhancement of ventilation in these patients took place at the expense of deepening of breathing, as compared to normal individuals with its normal frequency. In all these patients there was observed a reduction of vital capacity, maximum breathing capacity of the lungs, and the respiratory reserve volume.

The spiograms showed a uniform and regular breathing of considerable depth; this character of breathing was retained upon repeated spirometry carried out within two months after the first spiogram in patient V., and within eight months -- in patient I.

Upon transition to oxygen respiration, there was observed in these three patients a reduction of the minute volume of respiration. Our data do not correspond to the data of Knipping, etc.

Of different character are the data of patient B. P. This is the only one of the four who had complained of dyspnea; he noted, however, that he is capable of performing heavy physical work without dyspnea, whereas outside of work the dyspnea appeared in the form of periodic deep inspirations.

We could observe this character of breathing also during our routine conversation with this patient; according to the statements of his kin, the patient's breathing became regular only during sleep. Outside of some deep inspirations, the breathing of this patient was more frequent and superficial; the spirometry showed that against the background of deep inspirations of 250 to 400 ml, after each 10 to 15 respirations, there appeared an inspiration of the length of two-and-a-half to three normal inspirations of cycle and depth of two to two-and-a-half liters. This character of the spirogram was also observed in the patient in an examination repeated a year later.

In spite of his complaints of dyspnea, the indices of vital capacity and maximum breathing capacity of the lungs in this patient were normal.

The hyperventilation syndrome can thus proceed differently, in correspondence with the variety of clinical course of neuroses, and it can manifest itself variously upon examination of the function of external respiration. The common symptom is a marked increase of ventilation during rest, observed in the form of a uniform deep breathing, or in frequent breathing of normal depth with periodic deep inspirations. The hyperventilation syndrome represents the result of cortical impairment of respiration in a neurasthenic type of neurosis. It can be observed in patients suffering from neuroses and, at times, be the cause of incorrect diagnosis of cardiovascular, pulmonary, intestinal and other internal diseases.

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